

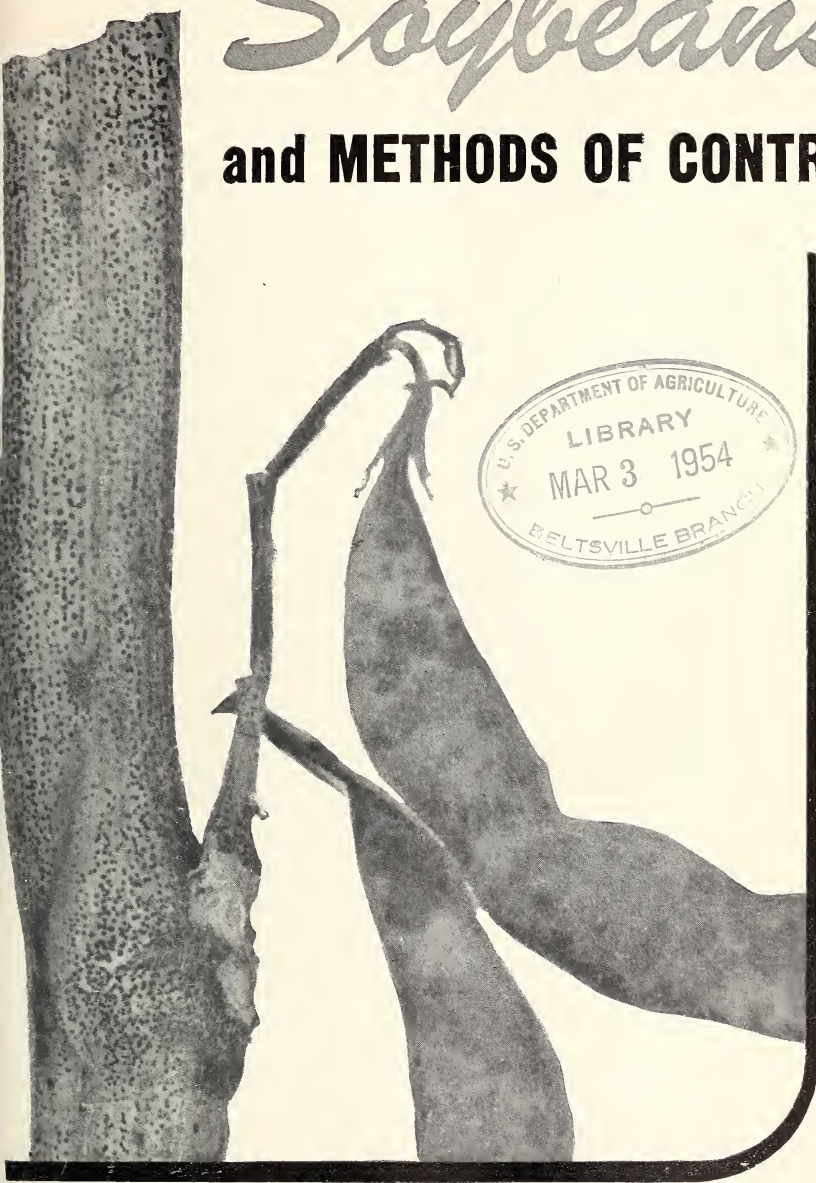
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DISEASES OF

Soybeans

and METHODS OF CONTROL



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Diseases of Soybeans and Methods of Control¹

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Approximately 50 diseases are known to occur on soybeans in the United States (81).³ About half of these occur commonly each year in one or more of the major producing areas, and the total loss they cause is great. Consequently, research on soybean diseases is being conducted

in an attempt to develop effective control measures for the more prevalent and important diseases. Much of this work is in cooperation with plant breeders who are developing new, improved varieties of soybeans that are resistant to one or more of the major diseases.

¹ Cooperative investigations of the former Bureau of Plant Industry, Soils, and Agricultural Engineering and the agricultural experiment stations of Mississippi, Illinois, and North Carolina.

² Daniel Guravich, formerly geneticist, Mississippi Agricultural Experiment Station, furnished the photographs for figures 9, 10, 11, 15, and 20, and Lewis W. Erdman, bacteriologist, of the Bureau, that for figure 25.

³ Italic numbers in parentheses refer to Literature Cited, p. 36.

The material in this Circular is published to aid soybean growers, county agents, extension workers, and agricultural research workers in identifying the diseases and to suggest such control measures as are now available. Since an annotated

bibliography of 503 titles on soybean diseases prepared by Ling (64) was published in 1951, no attempt has been made to have the Literature Cited section of this Circular all-inclusive.

LEAF, STEM, POD, AND SEED DISEASES

BACTERIAL BLIGHT

Bacterial blight is one of the most widespread diseases of soybeans. Although it may appear on the stems and pods, it is most common and conspicuous on the leaves. The first symptoms on the leaves are small, angular, yellow spots, frequently water-soaked at the center. Later, as the tissues die, the center of the spot becomes brown to black. It is surrounded by a water-soaked area that is bordered by a yellowish halo (fig. 1). Many small infec-

tions sometimes run together, causing large dead areas. Many of these dead areas fall out during wet weather. Under certain conditions the infection travels along the tissues next to the veins. At times this may result in badly shredded leaves, especially during periods of wind and rain. Heavy infection may cause defoliation. Bacterial blight is likely to be most serious during periods of cool weather and frequent rain. It is caused by *Pseudomonas glycinea* Coerper (16). The bacteria are seed-borne (45)



FIGURE 1.—Bacterial blight on the upper surface (A) of a soybean leaflet and on the lower surface (B).



FIGURE 2.—Bacterial pustule on the lower surface (A) of a soybean leaflet and on the upper surface (B).

and also can survive in dead leaves from one growing season to the next.

Susceptibility to the disease varies with soybean varieties. Flambeau and Hawkeye, for example, appear to be less susceptible than most other northern varieties, but they are not immune to the disease.

BACTERIAL PUSTULE

Bacterial pustule is present to some extent in most soybean-growing areas. In the Northern States its prevalence and severity seem to vary considerably with the season, but in the South it is more uniformly severe (32). The pods may

become infected, but the disease is most common on the leaves. The symptoms are much like those of bacterial blight. At first, they appear as small, yellowish-green spots with reddish-brown centers, more conspicuous on the upper surface of the leaf (fig. 2). A small, raised pustule usually develops at the center of the lesion, especially on the lower leaf surface. This is the stage at which the disease is most readily distinguished from bacterial blight. The pustule and the absence of water soaking serve to distinguish bacterial pustule from blight. In bacterial pustule, small infections may run together and cause large, irregular, brown areas,

surrounded by a yellow margin. Parts of the brown dead area may break out, giving the leaf a ragged appearance. This disease is caused by *Xanthomonas phaseoli* var. *sojense* (Hedges) Starr & Burk. (34). The bacteria that cause the disease overwinter in diseased leaves. According to work done at North Carolina and Iowa, the bacteria also are seed-borne.

Although most commercial varieties of soybeans are susceptible to bacterial pustule, the variety CNS is highly resistant (33). Ogden variety also has some resistance (24).

WILDFIRE

Wildfire, caused by the bacterium *Pseudomonas tabaci* (Wolf & Foster) Stevens (2), was first recognized as a disease of soybeans in 1943. It was observed in most of

the North and South Central States in 1944. In general, it is more serious in the South than in the North Central States.

Light-brown necrotic spots of variable size, surrounded by broad, yellow halos, appear on the leaves (fig. 3). At times the necrotic spots are dark brown to black, with rather indistinct halos. In damp weather the lesions enlarge and run together, forming large dead areas on the leaf. Such severe infection may cause considerable loss of leaves.

Several investigators have noted that wildfire infection is most prevalent when bacterial pustule is common. The presence of pustules at the center of wildfire lesions has led to the belief that wildfire infection takes place most readily when the leaves are infected already with pustule. Experiments in North Carolina and Illinois indicate that



FIGURE 3.—Wildfire, another bacterial disease of soybeans, is characterized by broad, yellow halos around the dead areas on the leaflets.

most wildfire infection takes place through the bacterial pustule lesions and that bacterial blight lesions may also serve the same purpose. Attempts to infect soybeans with the wildfire organism in the absence of wounds or bacterial disease lesions have usually been unsuccessful. Furthermore, the variety CNS, which is highly resistant to bacterial pustule, has remained generally free of wildfire infection in the field.

Tests in North Carolina show that the organism is seed-borne and can live for 3 to 4 months in infected leaves on the ground (31). In buried leaves, the organism lived less than 4 months. Seed stored for 18 months produced plants free of wildfire. Fall plowing might give some control of infection carried over in diseased leaves.

BROWN STEM ROT

Brown stem rot was first discovered in central Illinois in 1944. It is confined mostly to the Midwest, where it is a serious disease in Illinois, Indiana, Ohio, and Iowa (4). It occurs also in parts of Missouri, Minnesota, Kentucky, and Canada.

The fungus enters the plant through the roots and lower stem. The first symptoms appear in late July or early August. When infected stems are split open, a brown discoloration can be seen inside the lower part of the stem (fig. 4, A). Cool weather in August favors development of the disease, and eventually the browning becomes continuous throughout the stem. Sustained high temperatures in July and August suppress the disease.

Leaf symptoms are not evident in the early stages of the disease but appear in late August or early Sep-

tember. Sudden blighting and drying of the leaves take place so rapidly that the grower often suspects frost damage. The leaf tissues between the veins turn brown, while those near the veins remain green for several days (fig. 5). Soon the entire leaf withers. From a distance, a badly infected field appears brownish in contrast with the yellow green of a normally maturing field. Considerable lodging occurs in diseased fields, and the size of the seeds may be reduced.

One of the peculiarities of brown stem rot is the fact that leaf symptoms do not always occur. In some seasons infected plants do not develop these symptoms and the disease may be unnoticed, unless the stems are split open for examination. Observations indicate that leaf symptoms occur when a week or two of cool weather in August is followed by a warm, dry period, during which time the stems cannot transmit an adequate supply of water.

Brown stem rot is caused by the fungus *Cephalosporium gregatum* Allington & Chamberlain (4). It is soil-borne. There is no evidence that it is carried on the seed. The only control measure known at present is a rotation wherein soybeans are grown on a field only once in 3 or 4 years.

POD AND STEM BLIGHT

Pod and stem blight, caused by *Diaporthe phaseolorum* (Cke. & Ell.) Sacc. var. *sojae* (Lehman) Wehm., is chiefly a disease of the stems and pods of plants nearing maturity (51, 65). It can be identified by the numerous, small, black fruiting bodies (pycnidia) that appear on the stems and pods of in-

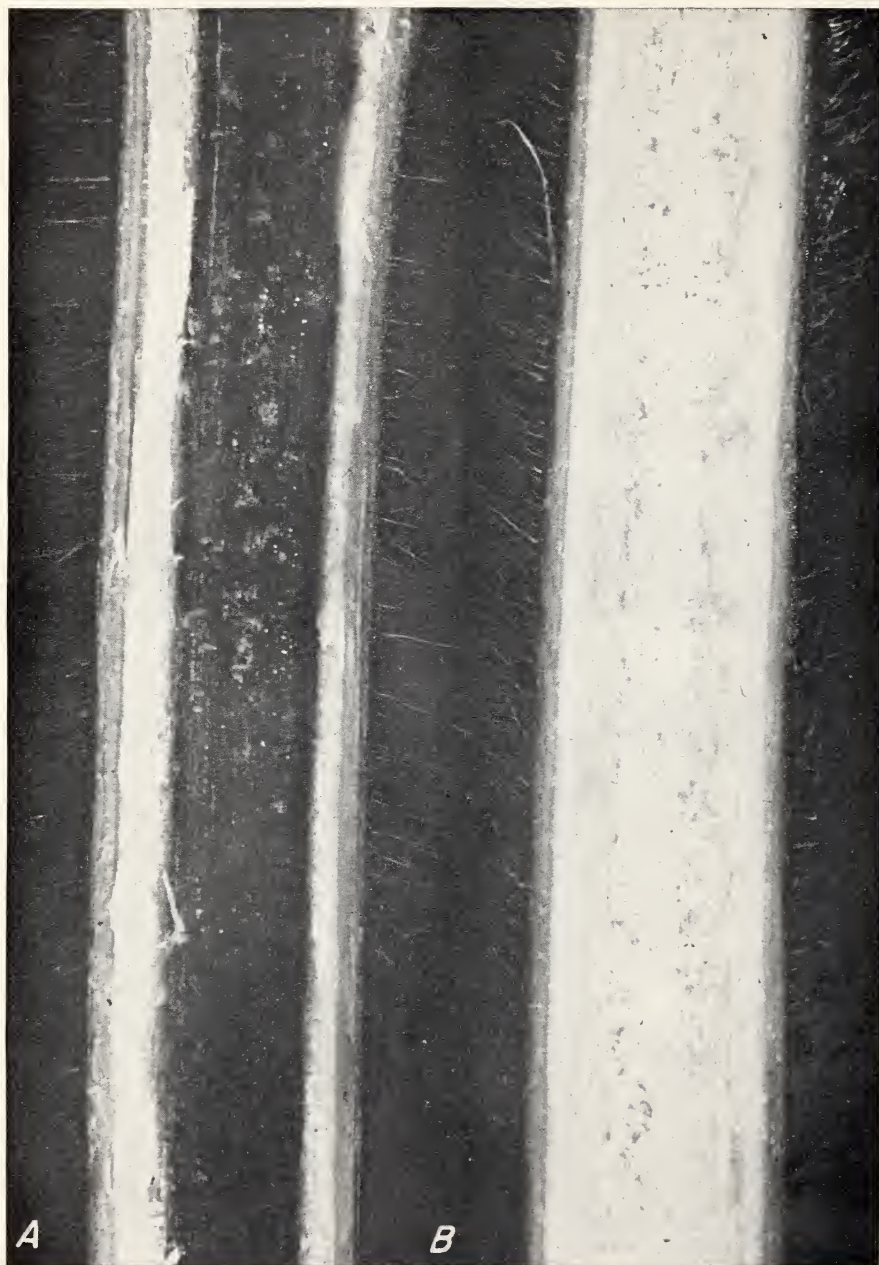


FIGURE 4.—Soybean stems split open to show (A) the dark-brown internal discoloration characteristic of brown stem rot and (B) the white center of a normal stem.



FIGURE 5.—Brown stem rot symptoms appear on the soybean leaflets in late August or early September as a sudden blighting.

fectured plants. On the pods the pycnidia are scattered, while on the stems they usually are arranged in linear rows. Seedlings developing from infected seed are destroyed before, or soon after, emergence. The disease is well distributed over most of the United States.

High humidity and rain are conducive to the formation and spread of the spores from the pycnidia. The disease is seed-borne, and the fungus also can overwinter on diseased stems in the field. Consequently, complete plowing under of crop refuse, the use of disease-free seed, and crop rotation are recommended as control measures.

STEM CANKER

Stem canker and pod and stem blight were considered one disease for many years. In 1948 Welch and Gilman in Iowa (82) reported that two separate diseases were involved. They found that pod and stem blight attacked older plants nearing

maturity, while stem canker killed plants earlier in the season. Stem canker is caused by the fungus *Diaporthe phaseolorum* (Cke. & Ell.) Sacc. var. *bataatatis* (Harter & Field) Wehm.

Stem canker appears to be more common in the Midwest, especially in Illinois, Indiana, and Iowa, than in other regions. It usually appears after mid-July. Dead plants, with the dried leaves still attached, may be the first indication of its presence. A brown, slightly sunken type of lesion is found girdling the stem, usually at the base of a branch or leaf petiole (fig. 6). The stem canker fungus does not produce pycnidia on the living host, but sexual fruiting bodies (perithecia) develop on dead stems in the field during the winter. The spores (ascospores) produced in them serve to spread the disease during the following season. Timnick and co-workers (77) report the production of pycnidia and asexual spores in culture.



FIGURE 6.—Stem canker lesion girdling a soybean stem at base of branch on the right.

Stem canker, like pod and stem blight, is seed-borne. Since dis-

eased stubble in the field is also a source of infection for both diseases, the same control measures are recommended as for pod and stem blight. As yet, there is no variety known to be resistant to stem canker.

FROG-EYE

The first report of the frog-eye disease of soybeans in the United States was from North Carolina in 1928 (52). The disease is now widely distributed on soybeans in the United States. It is primarily a foliage disease, but also occurs on stems, pods, and seeds (54).

On leaves the disease appears as a small "eyespot," with a brown, gray, or nearly white central area surrounded by a narrow, reddish-brown border. No chlorotic tissue appears between the border and the green color of the normal leaf (fig. 7). A dark-gray cluster of the



FIGURE 7.—Frog-eye lesions on the lower surface (A) of a soybean leaflet and on the upper surface (B).

sporophores of the causal fungus (*Cercospora sojina* Hara (*C. daizu* Miura)) is usually visible near the center of each spot and is useful for identification of this disease. Heavily spotted leaves fall prematurely, thus causing a loss to the hay and seed crop.

Infection occurs on stems and pods late in the growing season, but these are less conspicuous and less easily identified than those on leaves (54). Stem lesions are elongated and reddish with a black border when young. They become brown, then pale gray, and finally almost black when fruiting organs of the causal fungus develop on the surface. Infection on ripe pods appears as circular, brown to light-gray spots, usually encircled by a narrow dark ring. The fungus frequently grows through the pod wall into the seed (73). The causal fungus overwinters on debris left in the field from a previous diseased crop and is introduced into new fields through the planting of infected seed.

Seed treatment with chemicals does not give satisfactory control of frog-eye. Crop rotation aids in holding the disease in check. Soybeans should not be planted on land that grew a crop diseased with frog-eye the preceding year. Increased prevalence of this disease has been noted on susceptible varieties in some areas in recent years. Observations show that severity of the disease varies greatly among soybean varieties.

BROWN SPOT

Brown spot was recognized in the United States for the first time in North Carolina in 1922. Formerly

regarded as a disease of minor importance, brown spot has greatly increased in prevalence and severity in recent years. It sometimes causes serious defoliation and yield reduction.

The causal fungus is seed-borne and the disease appears early in the season on both surfaces of the primary leaves of young plants as small, reddish-brown, more or less angular spots (fig. 8). The spots vary in size up to about one-fifth inch and are usually surrounded by a zone of light-green or yellow tissue of indefinite width. Spores produced on these primary leaves spread upward and infect trifoliate leaves, stems, branches, and pods (83). As the plants approach maturity, numerous, small lesions appear and often coalesce to form large, irregular, brown, dead spots on the upper leaves. On heavily infected leaves much of the uninvaded tissue between the dead spots becomes chlorotic and greatly impaired in function. Numerous light-brown pycnidia, sometimes visible with the aid of a hand lens, occur in the dead tissue. Brown discolorations of irregular shape and size occur on stems and pods, but these cannot readily be distinguished macroscopically from infections caused by other fungi.

Brown spot is caused by the fungus *Septoria glycines* Hemmi. The fungus grows through the pod wall into the seeds. Lesions on the infected cotyledons furnish spores for subsequent infection of primary leaves. Seed from badly diseased fields should not be used for planting, as seed treatment does not give satisfactory control of this disease. The causal fungus remains viable during the winter on diseased stems



FIGURE 8.—Brown spot lesions on the primary leaves of young soybeans.

and leaves left in the field. Greatest damage occurs where soybeans are planted every year in the same field. When this practice is followed, severe brown spot infection starts early in the season and half or more of the foliage may be lost before maturity. Some other crop should be grown for at least 1 year between successive crops of soybeans. Varieties of soybeans differ considerably in respect to the amount of damage caused by the brown spot disease.

ANTHRACNOSE

Soybean plants in all stages of growth are subject to infection by anthracnose. When infected seeds are planted, many of the germinating seeds are killed before emergence. Dark-brown sunken cankers are often seen on the cotyledons of seedlings that do emerge. The causal fungus may completely destroy one or both cotyledons or grow from them into the tender tissues of the young stems. There, it produces numerous, small, shallow, elongated, reddish-brown spots or large, dark-brown, deep-seated lesions that usually kill the young plant. The causal fungus is cap-

able of growing within stem, leaf, and pod tissues of soybean plants without giving external evidence of its presence until some favorable condition of the plant induces sporulation of the fungus on the surface of the invaded tissue (76).

On older plants anthracnose reaches its most destructive stage of development during rainy periods in late summer. Lower branches, which are weakened by shading, are killed by the fungus. Likewise, young pods may be attacked. Indefinite brown areas develop and coalesce to cover the entire surface of the diseased stems and pods, and the causal fungus forms numerous, black fruiting bodies (acervuli) on the diseased areas (fig. 9). As these disease organisms develop, several short, dark spines (setae) emerge from each acervulus, giving a dark, stubble-bearded appearance to the diseased area. Seeds in diseased pods may be shriveled and moldy. On the other hand, infected seeds may exhibit no outward sign of disease.

Anthracnose of soybean is caused by two species of fungi, each producing similar symptoms. *Gloemerella glycines* (Hori) Lehman & Wolf was first reported as the

causal agent of soybean anthracnose in America (63). The imperfect fungus (*Colletotrichum truncatum* (Schw.) Andrus & W. D. Moore) that causes stem anthracnose of lima bean is now widely distributed on soybean and is probably the fungus most commonly found associated with soybean anthracnose (39). Both fungi survive from one growing season to the next in diseased stems left in the field. Both overwinter in infected seeds and are spread by planting diseased seeds in new localities. Treatment of anthracnose-infected seed with fungicides greatly improves the stand, but it does not eliminate the disease. Rotation should be practiced to avoid outbreaks of disease that might arise from overwintered diseased stems.

TARGET SPOT

Target spot primarily affects the soybean leaf blades. However, the fungus also causes spotting of the leaf petioles, stems, pods, and seeds. On the leaf blades the spots are reddish brown, circular to irregular in shape, and vary from pinpoint size when immature to one-half inch or more in diameter. The larger spots are sometimes distinctly zonate, and the name "target spot" was suggested by this zonation (fig. 10). The spots of dead tissue are frequently surrounded by a dull-green or yellowish-green halo, thus resembling wildfire. However, target spot is reddish brown, and usually the larger spots are zonate. Narrow, elongated spots are sometimes observed along the veins on the upper leaf surface. On petioles and stems the spots are dark brown and vary from mere specks to elongated, spindle-shaped lesions. Pod spots



FIGURE 9.—The dark fruiting structures (acervuli) of an anthracnose fungus on a soybean stem.

are generally circular, about one-sixteenth inch in diameter, with slightly depressed purple-black centers and brown margins. The fungus penetrates the pod wall in some cases and causes a small, blackish-brown spot on the seed.

This disease is caused by the fungus *Corynespora cassicola* (Berk. & Curt.) Wei (*Helminthosporium vignicola* (Kawamura) L. Olive) (67, 79). It was first reported in the United States in 1945 (68) and is now prevalent on soybeans grown in the alluvial soil area adjacent to the Mississippi River in the States of Louisiana, Mississippi, and Arkansas. It is known to occur also on the Coastal



FIGURE 10.—Target spot lesions on soybean leaflets, showing zonation within the lesions and a narrow chlorotic halo around them.

Plain in North Carolina, South Carolina, Georgia, Florida, Alabama, and Mississippi, and in the hill section of Mississippi. It appears capable of becoming a serious disease of soybeans in the southern areas. So far, it has not been reported from the Corn Belt States.

Ogden, the soybean variety grown most commonly in the South, possesses moderate resistance to this disease. Soybean breeders and pathologists are on the alert to prevent the release of new soybean varieties more susceptible to this disease than Ogden. The fungus attacks cowpeas as well as soybeans. These two crops should not be grown in close proximity or in sequence in the rotation. Rotation and plowing under of crop refuse in the fall should aid in controlling target spot.

PHYLOSTICTA LEAF SPOT

Leaf spot caused by a species of *Phyllosticta* was first discovered in the United States in 1927 (75). In some years phyllosticta leaf spot has caused appreciable premature defoliation in localized areas, such as the Eastern Shore of Maryland (69) and southeastern Missouri (18).

The leaf spots are round to oval in shape, vary from light to dark brown, and are approximately one-fourth inch in diameter. Spots near the margin of the leaf blade frequently coalesce, giving the leaf margin a fired appearance (fig. 11). The small, black, fruiting dots of the fungus are found abundantly in the leaf spots. Severely attacked leaves fall from the plants prematurely. The fungus also causes le-

sions on the petioles and stems, which add to the total injury. On the Eastern Shore of Maryland in 1951, lesions on the soybean pods were observed and infected seed were found beneath the lesions.

This disease is caused by the fungus *Phyllosticta sojaecola* Massal. (*P. glycinea* Tehon & Daniels) (10). An ascospore - producing stage of this fungus (*Pleosphaerulina*) has been reported from the Orient, but so far it has not been observed in the United States. Plowing under the crop refuse in the fall should aid in controlling this disease by destroying much of the overwintering inoculum.

DOWNY MILDEW

The downy mildew disease of soybeans is characterized in its early stages by indefinite yellowish-green areas on the upper surface of the leaves. In severe cases entire leaflets are thus discolored. As infection progresses, the diseased areas become grayish-brown to dark-brown necrotic lesions surrounded by yellowish-green margins (fig. 12). Severely infected leaves fall prematurely. A grayish, moldlike growth develops on the undersurface of these lesions, and the spores produced on this growth spread the disease from plant to plant. In addition to these externally borne summer spores, thick-walled resting spores develop within the leaf tissues. These overwinter in the fallen leaves and probably provide inoculum for infecting the next season's crop.

In addition to these foliage symptoms, the causal fungus (*Peronospora manshurica* (Naoum.) Syd.) grows within the pods and covers

part of the seeds with a white crust composed largely of the thick-walled resting spores (fig. 13) (43). When such seeds are planted, a small percentage of the seedlings have the fungus growing within them (37, 44). The first leaves to unfold on such seedlings sometimes are covered with mildew growth and thus provide centers of infection in the new season's crop.

Downy mildew occurs throughout the soybean-growing areas of the United States and in some years is one of the most prevalent diseases. However, severe epidemics have not been observed, and the loss appears to be restricted to some degree to premature defoliation, depending on seasonal conditions. Experimental tests at the Wisconsin Agricultural Experiment Station have demonstrated the existence of three races of the parasite (28). Differences in susceptibility have been observed in soybean strains and varieties. Should the disease become more serious, resistant varieties probably could be developed.

POWDERY MILDEW

Powdery mildew may be recognized in its early stages by the presence of small colonies of thin, light-gray or white mycelium spreading rapidly on the upper surface of the leaf. Reddening of the underlying leaf tissue may or may not be evident. In time, the whitened areas of mycelium enlarge but seldom coalesce to cover all the leaf surface. Many conidia are produced, causing the infected areas to take on a white powdery appearance as though coated with flour (fig. 14). Heavily infected leaves soon become etiolated, turn brown, and drop off.



FIGURE 11.—*Phyllosticta* lesions on the primary leaves of young soybeans. The small, black dots on the lesions are the fruiting structures (pycnidia) of the fungus.

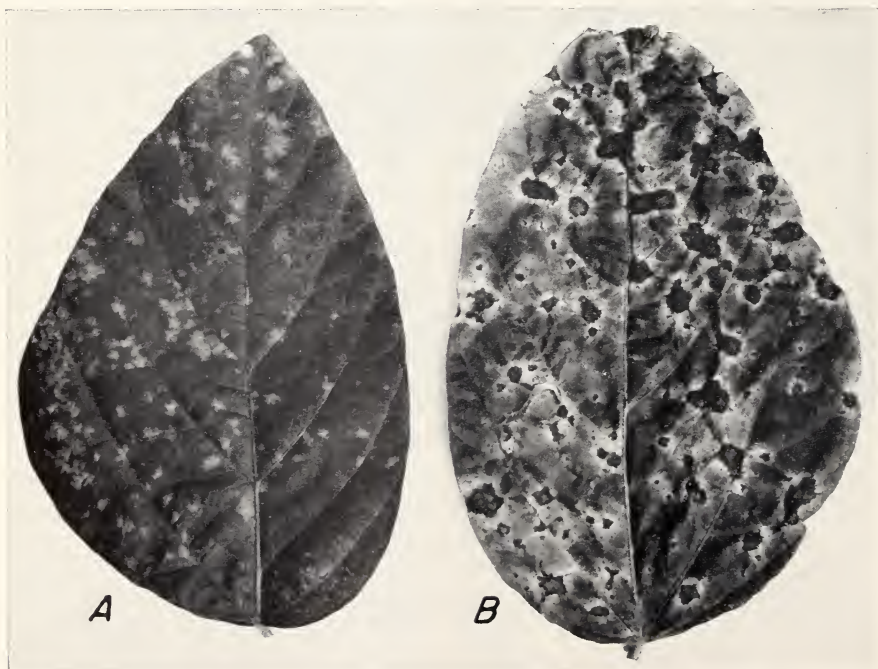


FIGURE 12.—Downy mildew lesions on upper surface of soybean leaflets, showing (A) early stage and (B) later stage of the disease.



FIGURE 13.—Soybean seeds encrusted with oöspores of the downy mildew fungus.

Leaf petioles and green stems are attacked in the same manner. Near the time for frost, large numbers of ascocarps develop on the diseased parts. These are white at first, then orange, and finally become dark brown or nearly black when fully mature.

Powdery mildew of soybean is caused by one or more species of fungi. Early accounts of the disease (53) give *Erysiphe polygoni* DC. as the name of the causal agent, largely on the assumption that the soybean fungus is the same species as occurs on garden beans and clover. However, specimens observed in recent years in the United States have had ascocarps with bifurcately

branched appendages and other morphological characters of *Microsphaera* (58). This fungus has been found on field-grown soybeans in North Carolina and South Dakota and on soybeans in the greenhouse in North Carolina and Illinois. It resembles, and probably is to be considered, a form of *Microsphaera diffusa* Cke. & Pk.

The Ogden variety, grown widely in the South, is susceptible, while Roanoke appears to be immune. The causal fungus spreads rapidly on susceptible varieties in the greenhouse. In the field it occurs only sporadically and does not appear to be adapted to prolonged survival.



FIGURE 14.—The powdery mildew fungus causes a powdery, flourlike coating on soybean leaflets.

BLACK PATCH

On soybean leaves the black patch fungus causes small, circular to irregular spots that are buff at the center and have narrow purplish borders. They resemble frog-eye leaf spot lesions, but the black patch disease can be recognized by the dark-brown to black aerial hyphal strands on the surface of the spots. This is a disease of minor importance, and so far it has been observed on soybeans only in Georgia (80). The fungus attacks clovers and other forage legumes rather commonly. In no instance has the fungus been observed to produce fruiting structures on its host or in culture and, consequently, it remains undetermined taxonomically.

ALTERNARIA LEAF SPOT

Alternaria leaf spot is a rather common disease of soybeans in

many parts of the United States, especially in the Midwest (13). Although occasional infection occurs on young plants, it is generally a leaf disease of plants nearing maturity and occurs too late to cause serious damage. The spots are brown to dark brown with concentric rings, and may vary from $\frac{1}{4}$ to 1 inch in diameter. The spots often coalesce, producing large necrotic areas on the leaf blade (fig. 15). A fungus of the genus *Alternaria* has been consistently found on these spots, and inoculations have shown that it can produce the disease (42). The species of the fungus has not been determined. *Alternaria* leaf spot resembles target spot, but it lacks the reddish-brown color and the yellowish-green halo that characterize target spot.



FIGURE 15.—*Alternaria* leaf spot, showing zonation of the spots and the dead areas that develop when spots coalesce.

Small, brick-red spots on soybean leaves have been investigated at the Arizona Agricultural Experiment Station. These spots, primarily, caused by sunburn or aphid injury, were found to enlarge, turn brown, and were frequently covered with a sooty-black fungus growth. The fungus, described as *Alternaria atrans* Gibson, was considered to be a weak parasite (30).

ARSENICAL INJURY

Soybean leaves are very sensitive to arsenical injury. This type of damage results in a brown leaf spot with concentric rings, very similar in appearance to the *alternaria* leaf spot. These spots, however, do not show the sooty-black fungus growth at first. Late in the season they may become overgrown by the *Alternaria* fungus.

Arsenical injury has been observed in Illinois after poison bait containing arsenic had been scat-

tered over the plants to kill grasshoppers (42), and in greenhouses where plants have been treated to control slugs or other pests. In the South, calcium arsenate dust applied to cotton for insect control may drift over soybean fields and cause spotting of the leaves. In severe cases of injury premature defoliation may result.

SOYBEAN MOSAIC

Clinton reported soybean mosaic from Connecticut in 1916 under the name of chlorosis, or crinkling (15). Gardner and Kendrick of the Indiana Agricultural Experiment Station established the virus nature of the disease (26) and Kendrick and Gardner (46) confirmed the fact that it is seed-transmitted. Heinze and Koehler, working in Germany, demonstrated in 1940 that aphids transmit the virus (35). This disease, caused by *Soja virus* 1, is found commonly in all of the

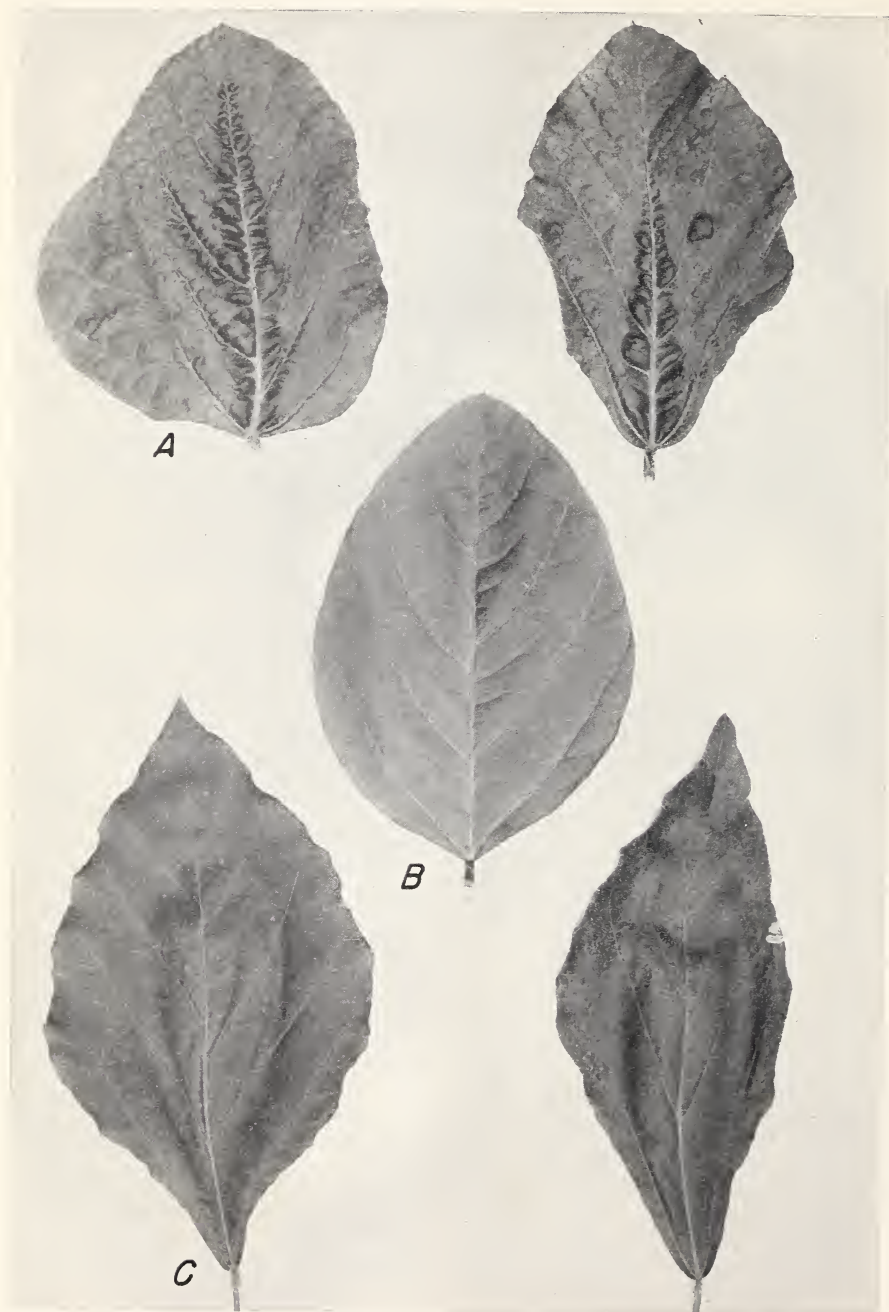


FIGURE 16.—Soybean leaflets, showing (A) mosaic symptoms (B) a normal leaflet, and (C) 2, 4-D injury.

soybean-producing areas of the United States.

The leaves become distorted and may be narrower than normal, with margins turning downward. Some varieties show a severe ruffling along the margins of the main veins (fig. 16, *A*), as compared with the smooth surface of a healthy leaf blade (fig. 16, *B*). Plants infected with mosaic virus frequently are stunted. This is especially true for some of the vegetable varieties, such as Bansei. On oil-type soybean varieties, temperature has a marked effect on leaf symptoms. Plants that have leaf distortion early in the season show less evidence of the disease with the onset of high summer temperatures. Some of the vegetable varieties, however, continue to develop leaf distortion, regardless of temperature. Plants affected by mosaic virus produce misshapen pods and fewer seeds than normal plants. Since the virus is seed-borne, diseased plants should be rogued from fields where soybeans are grown for planting seeds.

Leaf symptoms strongly resembling those caused by mosaic virus are frequently caused by the application of 2,4-D (fig. 16, *C*). The common use of this chemical in weed control has led to many false reports of widespread virus infection in soybean fields near the site of application. Spraying weeds with 2,4-D on windy days may result in injury to soybeans several hundred feet away. Most of this injury appears to be temporary, with only 3 or 4 leaves showing distortion, and the affected plants then recover. Whether this has any effect on yield has not been established.

YELLOW MOSAIC

Conover (17), in Illinois in 1948, found that what had been called soybean mosaic actually consisted of two diseases, mosaic and yellow mosaic. Yellow mosaic of soybean is caused by *Phaseolus virus* 2, the same virus that causes the yellow mosaic of garden bean. The younger leaves of infected plants show a yellow mottling, scattered in random spots over the leaflets, or sometimes an indefinite yellow band along the major veins (fig. 17). Rusty necrotic spots develop in the yellowed areas as the leaves mature. Infected plants are not noticeably stunted.

There is no evidence that the disease is seed-borne. Its effect on yield is not serious. The disease is widely distributed in the Midwest, but infection thus far has not exceeded 1 percent in any field. It apparently is not common in the South.

BUD BLIGHT

Bud blight is caused by the most destructive virus of soybeans recognized to date. It was reported from Indiana in 1941 (70) and is distributed throughout the soybean-producing area of midwestern United States and in Ontario, Canada. It occurs rarely in the South. In the years between 1943 and 1947, losses ranged from 25 to 100 percent in some fields in the Midwest. Since then, infection by the bud blight virus has not been so prevalent or so severe.

The symptoms of bud blight are variable. When young plants are infected before blossoming, the tip bud turns brown, curves markedly,



FIGURE 17.—Yellow mosaic mottling on soybean leaflets.

and becomes dry and brittle (fig. 18). Often the leaf immediately below the tip bud shows a rusty flecking. The plant is dwarfed and produces no seed. There is sometimes a browning of the pith inside the stem in the region of the nodes below the blighted terminal bud.

If infection takes place about blossoming time, the plant may produce small, undeveloped pods, or no pods at all. Infection later in the season may result in the formation of poorly filled pods or pods that show a conspicuous purple blotching. Many of these pods drop prematurely. Plants infected with bud blight virus remain green after normal plants have matured and are thus easily found in the fall.

Bud blight is caused by the tobacco ring spot virus (3). There is

no definite evidence that the disease is seed-borne. The disease usually appears first at the border of a field, progressing inward. This suggests an insect carrier, but such an insect has never been found. In 1952 Walters, at Nebraska, demonstrated that grasshoppers can carry this virus from tobacco to tobacco (78). Whether this holds true for soybean is not known.

There is no known resistance to the bud blight virus in any soybean variety, and no effective control. Because of its direct effect on yield, it is potentially one of the most serious soybean diseases.

MINERAL DEFICIENCIES

Chlorosis of soybean leaves may be caused by a deficiency of mineral



FIGURE 18.—Recurved stem tip of a young soybean plant infected with "bud blight," a virus disease.

nutrients as well as by parasitic infections (19, 22). A chlorosis and firing of soybean leaves caused by insufficient potash is found rather frequently. The first sign of this deficiency is an irregular yellow mottling around the edges of the leaflets. The chlorotic areas soon merge to form a continuous yellow border. This is followed by marginal browning that may spread to include half or more of the area of the leaflet. Only the center and base of the leaflet may remain green when the deficiency is severe. The dead tissue usually falls out, giving the leaflets a ragged appearance.

Iron-deficiency chlorosis is characterized by large yellowish-green and then yellow areas between the veins. The entire leaflet, including

the veins and midrib, finally becomes involved as the green color disappears. This deficiency occurs only in certain districts where the soils are high in lime.

The leaf symptoms of manganese deficiency are quite similar to those of iron deficiency, and it is difficult to distinguish these two deficiencies except by chemical tests. Manganese deficiency has been observed in a few districts only.

Symptoms of iron and manganese deficiency first occur on the youngest leaflets as they are unfolding. Potash-deficiency symptoms, on the other hand, occur on the fully developed leaves.

Nitrogen deficiency is indicated by a gradual paling of the green color over the entire leaf blade, with only a slight yellowish tinge. Formerly, such symptoms were seen when noninoculated soybean seed was sown. They are not so common now, because most of the soils in the major producing areas have been inoculated with soybean root-nodule bacteria.

PURPLE SEED STAIN

The purple seed stain disease was reported on soybeans in the United States for the first time in 1927. This report (25) from the Indiana Agricultural Experiment Station called attention to the objectionable nature of the discoloration from the standpoint of the producer of pure seed. The disease has since been observed in most other States where soybeans are grown. Weather conditions prevailing at the time the seeds mature apparently have a pronounced influence on the percentage of discolored seeds that develop, since this varies widely from year to year with a given variety. Under

conditions favorable for the disease, 50 percent or more of the seeds of certain varieties may be discolored.

Although the symptoms of purple stain are most evident on the seeds, the causal fungus also attacks the leaves, stems, and pods. On the seed the discoloration varies from pink or light purple to dark purple, and ranges in size from a small spot to the entire area of the seed coat (fig. 19). Cracks often occur in the discolored areas, giving the seed a rough, dull appearance.

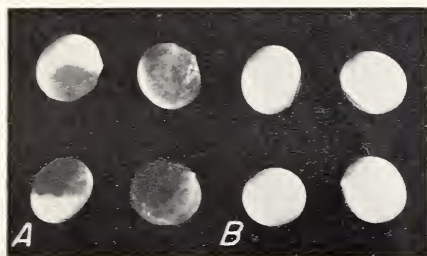


FIGURE 19.—Soybean seeds, showing (A) purple stain and (B) normal seeds.

When diseased seeds are planted, the fungus grows from the seed coats into the cotyledons and from these into the hypocotyl of a small percentage of the seedlings. The fungus produces spores abundantly on the diseased seedlings, and the wind blown and rain-splashed spores lodge on the leaves of adjacent plants. The leaf spots caused by these spores soon produce a second crop of spores that cause infections on other leaves, stems, and pods (66).

This disease is caused by the fungus *Cercospora kikuchii* (T. Matsu. & Tomoyasu) Gardner. The fungus probably survives the winter on diseased leaves and stems as well as in infected seed. Varietal differences in susceptibility have been ob-

served, and breeding for resistance to purple stain appears possible. Seed treatment aids in preventing loss of seedlings, but it will not assure freedom from purple stain in the seed crop. Dusting with copper dusts during the growing season has given partial, but inadequate, control of the purple stain disease in tests conducted at the North Carolina Experiment Station (59).

YEAST SPOT

Yeast spot was observed on seeds in the United States as early as 1928. In 1943 the disease was reported as occurring in several localities on a number of varieties in North Carolina (55) and on 25 varieties of oil and vegetable types of soybeans in Oklahoma (50). It also has been reported on soybeans in Virginia and South Carolina.

On the developing seed the infected tissue is slightly depressed and varies from yellowish or light cream to brown. There may be little or a considerable volume of dead tissue extending well into the infected embryo. On the surface of the dry seed coat the lesion appears as a very small discolored puncture.

This disease is caused by one of the group of yeast fungi, *Nematospora coryli* Pegl. (*N. phaseoli* Wingard). Apparently spores of the causal fungus are carried on the mouth parts of the green stink bug and are introduced into the developing seeds during the feeding activity of this insect. Seeds infected early may not mature or they may remain small and shriveled. Externally, pods may or may not show small, pinpoint, discolored areas in contact with the infected seeds.

ROOT AND CROWN DISEASES

CHARCOAL ROT

Charcoal rot is a disease of the roots and the basal portion of the stem of the soybean plant. When the bark is peeled from the roots and stem base, small, black specks—the sclerotia or propagating bodies of the causal fungus—may be seen (fig. 20, *A*). These are frequently abundant enough to impart a grayish-black color to the tissues beneath the bark, and this color has suggested the name “charcoal rot” for the disease. Occasionally, the fungus produces spore-bearing structures (pycnidia) in lesions on the soybean stem, and from this stage the fungus has been identified as *Macrophomina phaseoli* (Maub.) Ashby. This fungus appears to be a rather weak parasite of soybeans and attacks young plants only when their growth is retarded by hot, dry weather, poor soil, or some other unfavorable condition (13). The fungus is widely distributed in soils in the warmer sections of the United States and attacks other cultivated plants and weeds, as well as soybeans.

SCLEROTIAL BLIGHT

Sclerotial blight, like charcoal rot, is characterized by a rot at the base of the plant stem, but differs from charcoal rot in that the fungus sclerotia are larger and rounder and are brown instead of black. Furthermore, they are produced on a cottony, mycelial growth on the outside of the stem, rather than under the bark (fig. 20, *B*). Attacked plants die prematurely, sometimes before the seed has formed. The

disease is found in the sandy soil areas of the South where high summer temperatures occur; the name “southern blight” is sometimes applied to this disease. In some fields losses may be as high as 25 to 30 percent of the plants, but it is more common to see small, scattered areas of killed plants among the healthy ones.

Sclerotial blight is caused by the fungus *Sclerotium rolfsii* Sacc. The fungus attacks a wide variety of plants, including practically all the summer legumes adapted to the South (23, 36). Attempts to select soybean plants resistant to sclerotial blight at the Georgia Agricultural Experiment Station have been unsuccessful.

In North Carolina this fungus has been observed to cause circular, brown or straw-colored spots on the leaves (61). These spots usually have concentric markings and narrow, dark-colored borders of necrotic tissue (fig. 21). A clump of white mycelium or a small, spherical, brown sclerotium is often present at the center of the lesion. The size of the spot may vary from less than one-fourth to more than one-half inch in diameter. Leaf infections occur after wind and rain have splashed soil containing the causal fungus on the leaves.

STEM ROT

Stem rot, caused by the fungus *Sclerotinia sclerotiorum* (Lib.) DBy., also appears on the stem of the plant near the soil level. The fungus first forms a white cottony growth on the stem of the infected plant. Then, large black sclerotia



FIGURE 20.—A, Soybean stem base and root with bark peeled off to show the small, black sclerotia of the charcoal rot fungus; B, soybean stem showing white, immature and dark-brown, mature sclerotia of the sclerotial blight fungus.



FIGURE 21.—Lesions on soybean leaflets caused by the sclerotial blight fungus.

of various shapes are formed on the stem, partially covered by the white fungus growth. Sometimes the sclerotia form inside the stem and pods (13). These sclerotia are much larger than those of charcoal rot. They are black and irregularly shaped, thus differing from the round, brown sclerotia of sclerotial blight. The affected plants are killed by the girdling of the stem by the fungus.

The disease has been reported from Illinois, Iowa, Maryland, New York, and New Hampshire, and from Ontario, Canada. It is generally considered a minor disease, but has been locally destructive in a few fields. One field in Illinois in 1950 showed 10 percent of the plants killed. Koch and Hildebrand reported almost complete destruction of 3 to 4 acres of soybeans in a 12-acre field in Ontario in 1946 (1).

FUSARIUM BLIGHT, OR WILT

Fusarium blight, or wilt, occurs on sandy soils in the Southern States, but it has never caused as extensive losses in soybeans as have the similar wilts of cotton, cowpea,

and watermelon. Wilting is not a prominent symptom on soybeans, but the leaves of attacked plants yellow and fall off prematurely and the plants then die (20, 21). When the stem base and taproot of an attacked plant are split longitudinally, a brown or black discoloration of the vascular tissues is evident, as in the case of other diseases of this type caused by species of *Fusarium*. Experiments at the South Carolina Agricultural Experiment Station, reported in 1950, show that one or more races of *Fusarium oxysporum* Schlecht. f. *tracheiphilum* (E. F. Sm.) Snyder & Hansen can cause these symptoms on soybeans (6). In 1951 workers in South Carolina reported that the crotonaria wilt fungus (*Fusarium udum* Butl. f. *crotonariae* Padwick) also can infect certain varieties of soybeans and cause wilt (7).

Most varieties of soybeans now grown in the South appear to be resistant to fusarium wilt. Therefore this disease should not become one of major importance, unless newly released varieties prove susceptible or more virulent races of the fungus develop.

A root rot of soybeans caused by a *Fusarium* sp. has been observed in Illinois and Wisconsin. This fungus is probably different from those reported on soybeans in the South.

PYTHIUM ROOT ROT, SEED DECAY, AND DAMPING-OFF

Pythium root rot may occur on soybeans at any time from the young-plant stage to the end of the flowering period. It usually develops during, or soon after, protracted rainy periods. The diseased plants

stand singly or in small groups, and the leaves have the wilted or blighted appearance of plants affected with sclerotial blight. Cortical, or outer, tissues of the stem for some distance above the soil level are congested with water and appear translucent in the early stages of infection. Later, these tissues die, become brown, and the softer parts may fall out, giving a shredded appearance to the stem. The roots are usually so badly decayed that most of them break off when diseased plants are pulled from the soil. The outer tissues of the larger roots are brown, soft, wet, and slough off easily, exposing the central woody cylinder.

Pythium root rot is caused by one soil-inhabiting fungus (*Pythium ultimum* Trow) primarily (38), but other species such as *Pythium debaryanum* Hesse also may be involved (62). When soybean seeds are planted in cold soil, these fungi cause seed decay and destruction of very young seedlings before they emerge from the soil. A considerable proportion of the seedlings that do emerge exhibit the condition called "baldhead," having well-developed cotyledons but no plumule from which the main stem normally develops. Damping-off results from infection of the tender stems of young emerged seedlings. Thus, when soybeans germinate in cold soil, the stands may be greatly reduced by seed decay and damping-off. Losses from these phases of pythium root rot are much greater when the seeds used for planting are weak or low in vigor than when better seeds are used.

Stand losses from seed rot and damping-off are greatly reduced by seed treatment with protectants.

Provision for better drainage should reduce losses from pythium root rot of older plants.

RHIZOCTONIA ROOT ROT

Rhizoctonia root rot of soybeans (caused by *Rhizoctonia solani* Kuehn) usually occurs early in the growing season in the Midwest (11). It attacks young plants when the soil is unusually wet. The fungus causes a reddish-brown decay of the cortical layer of the main root and basal stem and destroys much of the secondary root system (fig. 22). The plants wilt and die. Dead plants usually appear in areas 4 to 10 feet in diameter, distributed irregularly over the field. The disease is important only in wet seasons. In 1950 and 1951, for example, when unusually frequent rains fell throughout July, rhizoctonia root and basal stem rot was found up to mid-August in the Midwest. As the soil dries out, infected plants die or show wilting at midday. When plants are pulled and examined, the basal portion of the taproot with its secondary roots are usually dead. Frequently, such plants have developed new roots just below the soil line and have partially recovered from the injury. As the upper layer of soil dries out, the plants can no longer obtain sufficient water with these shallow roots.

Rhizoctonia solani is a common soil fungus and causes root rot of a wide variety of crop plants. On soybeans it is generally a minor disease, but in wet seasons it may cause up to 10 percent loss in stand. In Minnesota seed treatment has given some promise of controlling damping-off and root rot, especially



FIGURE 22.—*Rhizoctonia* root rot of young soybeans, showing destruction of the secondary roots.



FIGURE 23.—*Rhizoctonia* aerial blight, showing (A) large, discolored areas on the leaf blades and (B) sclerotia of the fungus on a leaf petiole.

when the fungicide is pelleted on the seed (29).

When soybeans are grown in warm, humid areas, *Rhizoctonia* sometimes attacks the aerial portions of the plant and causes a spotting and blighting of the leaf blades. The spots or blighted areas are irregular in shape and buff to light brown (fig. 23, A). The causal fungus also grows over the leaf petioles and may form dark-brown sclerotia on these structures (fig. 23, B).

This disease has been observed in soybean fields in eastern North Carolina and occurs rather regularly on soybeans at Baton Rouge, La. So far, it has been of minor importance. Differences in varietal susceptibility have been observed in test plantings at Baton Rouge, but selecting or breeding strains of soybeans resistant to aerial blight has not yet seemed necessary. The fungus observed in North Carolina usually has produced sclerotia of the *R. solani* type. In Louisiana,

smaller sclerotia of the *R. microsclerotia* type are observed more commonly (9).

PHYMATOTRICHUM ROOT ROT

Phymatotrichum root rot of soybeans is caused by the same fungus (*Phymatotrichum omnivorum* (Shear) Dug.) that attacks cotton. It is prevalent in the heavy alkaline soils that occur in parts of Texas. Soybeans grown in this area are attacked by the fungus, causing the plants to wilt rapidly and die in patches of considerable size. If such plants are pulled from the soil, the bark of the roots is decayed. Frequently, brownish woolly strands of fungus growth can be seen on the roots. The fungus may persist in the soil for many years in its resting, or sclerotial, stage. Plants belonging to the grass family, such as corn, wheat, oats, and sorghum, are immune to this disease, and rotation with these crops is recommended to lessen the losses caused by this root rot. Plowing under of a green cover crop of sweetclover also has been found to lessen the severity of the disease in succeeding crops.

ROOT KNOT

The root knot disease occurs on soybeans growing in soil infested by parasitic nematodes of the genus *Meloidogyne*.⁴ Four species and one variety of this genus are able to attack soybeans, but probably the species most frequently found is *M. incognita* (Kofoed & White) Chitwood. The wormlike larvae of the

nematodes enter young roots, take permanent positions in the cortical tissue, and feed on nutrients supplied by the vascular cylinder. Cells adjacent to the developing female nematodes in the root are stimulated to greatly increased growth by secretions from the body of the parasite. Thus, knotlike swellings, commonly called galls, appear in the roots (fig. 24). Nematode galls cannot be removed without breaking the root. By this test they can be distinguished from the beneficial legume bacterial nodules that are weakly attached to the side of the soybean root and are easily broken away (fig. 25).

In a favorable host the female nematodes produce large numbers of eggs from which larvae develop and migrate to new positions in the root, thus producing more or bigger galls. In roots growing in a warm soil this process may be repeated every 30 or 40 days, thus rapidly increasing the nematode population and causing malformation of the roots of susceptible varieties.

The galls produced by the nematode species *M. incognita* are roughly spherical or elongate in shape and vary greatly in size, some being small swellings of the root, others as large as pecans. When infection is severe the roots have a clubbed appearance. Root galls caused by the species *M. hapla* remain small, and clusters of short adventitious roots emerge just above the galls and partially cover them. Soybean roots severely infected by this species are often densely matted, as a

⁴The genus *Meloidogyne* Goeldi was established in 1949 as the correct name for the root-knot nematodes formerly grouped under the name *Heterodera marioni* (Cornu) Goodey. At the same time it was shown that there are a number of different species of root-knot nematodes. They may be distinguished by morphological characters and host preferences (14, 71).



FIGURE 24.—Nematode galls (root knot) on soybean roots.

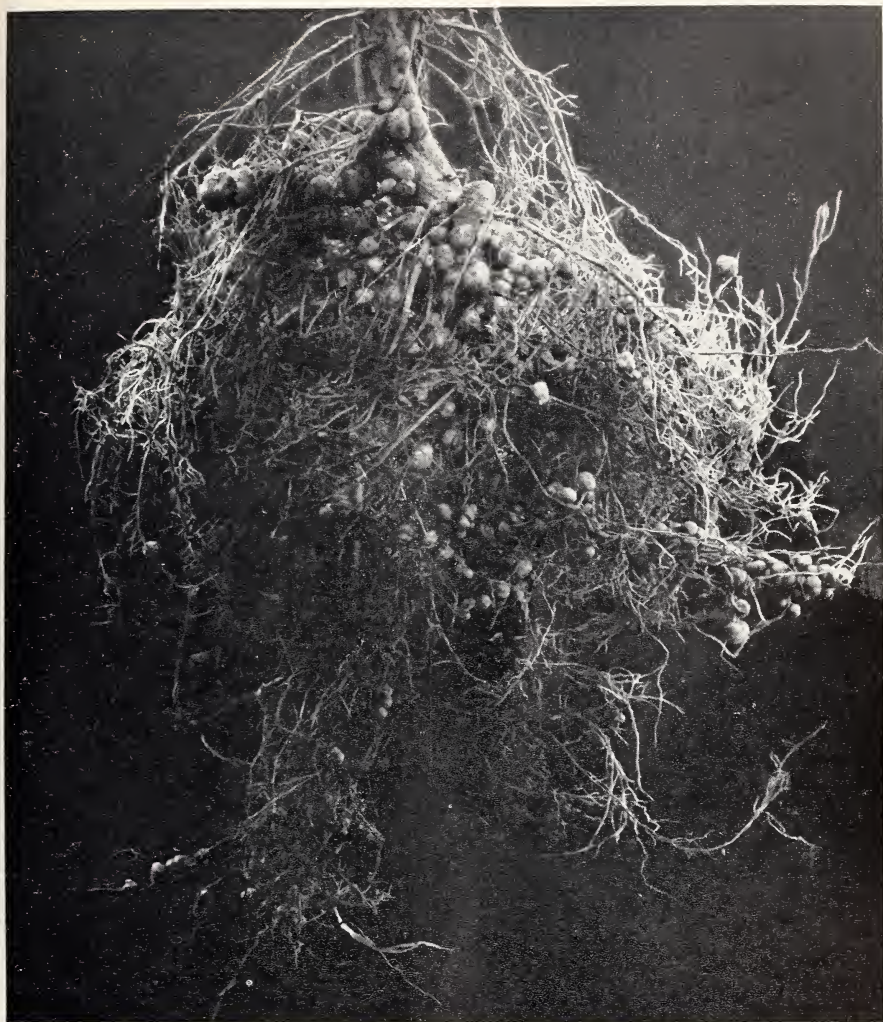


FIGURE 25.—Beneficial root nodules on soybean roots.

result of extensive root growth above the galls.

Symptoms on above-ground parts of affected plants vary from a slightly lighter green color to yellowing and marginal firing of the leaves. Slight or severe stunting may occur. During dry weather plants with galled roots wilt before normal plants, and many die a week or more before the normal date of

maturity. Yields of seed are often greatly reduced by root knot. Most varieties of soybean appear to be fairly tolerant of infection by the root-knot nematodes; consequently, the appearance of the parts above ground is not a good indication of the extent of disease on the roots. Plants having a liberal supply of soil nutrients and water may show little or no effect above ground,

even though the roots are heavily galled.

Control of root knot on soybeans is best obtained through use of resistant varieties. Laredo has long been recommended for planting on soil infested with nematodes; however, this variety is not resistant to root knot at all locations. Palmetto has been found to be the most resistant of the named varieties tested in North Carolina, and S-100 has been a close second. Armredo was found to be resistant in Arizona. The species of *Meloidogyne* involved in these tests has not been determined, however. When resistant varieties desirable for growth in a given location are not available, control of root knot can be obtained by reduction of the nematode population through use of other immune or highly resistant crops such as oats, rye, and crota-laria between successive crops of soybeans. Peanuts are resistant to *M. incognita*, *M. incognita* var. *acrita*, and *M. javanica* but are susceptible to *M. hapla* and *M. arenaria*. Thus, peanuts may be used as a resistant crop on soil infested

by *M. incognita*. *M. incognita* var. *acrita*, and *M. javanica* but should not be used when *M. hapla* or *M. arenaria* are present (71). The nematode population of the soil can be greatly reduced and significant damage from root knot prevented by soil fumigation, but this practice is economically feasible only where crops making high monetary returns per acre are grown.

LIGHTNING INJURY

Lightning striking in a soybean field may cause a circular spot of dead and injured plants that might easily be mistaken for the result of attack by some parasitic disease. If the plants are young, the affected areas may be 40 or 50 feet in diameter. When the plants are older and approaching maximum size, the lightning spots are usually much smaller. The clearly defined margins of the affected spots and the lack of any signs of a parasite being present will usually enable one to differentiate such injury from a parasitic disease, in spite of the similarity of the symptoms.

CONTROL MEASURES

DISEASE-RESISTANT VARIETIES

The most effective and economical measure for control of diseases of field crops is the use of disease-resistant varieties. In the older, established crops, such as wheat, oats, corn, and cotton, such varieties have been in use for many years. The soybean, being a relatively new major crop in the United States, is still in its infancy with respect to disease resistance. Consequently, one of the major problems at present is the finding of resistant types

for use in the soybean-breeding programs. Since 1943 the Department of Agriculture, in cooperation with several State agricultural experiment stations, has conducted a search for disease resistance in soybeans. Unfortunately, existing varieties appear to have little resistance to some of the more serious diseases.

A notable instance of progress in search of disease resistance was the discovery in North Carolina in 1943 that the variety CNS is highly

resistant to bacterial pustule. Although this variety lacks some of the better agronomic characters, its resistance to pustule makes it a valuable parent for hybridization with other varieties. Inheritance studies of crosses involving CNS have been reported by Feaster (24) and Hartwig and Lehman (33).

Lines derived from hybridization of CNS with superior varieties, such as Roanoke and Ogden, have yielded strains with the same high type of resistance to bacterial pustule as the CNS parent. Hybrids have also been made using Lincoln in the cross with CNS. These hybrid strains give excellent promise of future varieties combining the pustule resistance of CNS with more desirable compositional and agronomic characters, such as high oil content, resistance to lodging, and high yields.

About 1,200 varieties and plant introductions of soybeans were tested for resistance to bacterial blight in Illinois in 1947. Two of these, showing high resistance to the disease, were selected and used in crosses with locally adapted strains (12). Although still in the preliminary stages of testing, strains resulting from these matings show a high type of resistance to bacterial blight. While none of the standard varieties used in the Midwest are highly resistant to bacterial blight, there is considerable varietal difference in susceptibility. Illini, Lincoln, Blackhawk, and Monroe are highly susceptible; Richland and Hawkeye are intermediate; Flambeau and Patoka appear to have some resistance but develop the disease to a limited extent.

Workers at Wisconsin reported in 1946 that Mandarin No. 7, Ottawa Mandarin, Habaro, Dunfield,

Mukden, Flambeau, Kabott, Pride-soy, and several Manchu selections were resistant to downy mildew, while Richland and Illini were highly susceptible. Further work at the same station in 1950 showed that there were three physiologic races of the causal fungus (28). The variety Illini was found to be highly susceptible to all three races, Richland susceptible to two, while Chief, Manchu 3, Mukden, and Dunfield were resistant to all three races. Studies of the inheritance of resistance of soybeans to downy mildew have been reported also from Wisconsin (27).

Most, if not all, of the existing varieties are susceptible to rhizoctonia root rot. Reselection from the varieties Ottawa Mandarin and Flambeau and from the progenies of certain hybrids yielded 12 lines with a high degree of resistance to rhizoctonia root rot at the Minnesota Agricultural Experiment Station (49).

The severity of frog-eye leaf spot on certain varieties has been observed in Indiana in recent years, and studies of varietal resistance and of the inheritance of resistance have been undertaken (8). Experimental tests conducted in that State have shown that the varieties Capital, Flambeau, Ottawa, Mandarin, Blackhawk, Earlyana, Habaro, Monroe, Richland, Harosoy, Cypress No. 1, Dunfield, Illini, Gibson, Patoka, Hawkeye, and Chief are susceptible to frog-eye leaf spot, while Adams, Lincoln, Anderson, and Wabash are resistant. In the Southern States, most varieties grown for seed are resistant or only slightly susceptible, while some of the hay varieties, such as Tanner and Laredo, are very susceptible.

Resistance is still unknown for some of the most serious soybean diseases, such as brown stem rot, stem canker, and bud blight. Until disease-resistant varieties are available, the following practices are recommended to soybean growers as disease-control measures: (1) Crop rotation; (2) fall plowing under of the crop residues; (3) the selection of seed for planting from disease-free fields; and (4) seed treatment in certain areas.

CROP ROTATION

The parasites causing a number of soybean diseases are known to overwinter on or in diseased leaves and stems on the soil surface, and some also overwinter in the soil. Continuous cropping with susceptible varieties of soybeans would tend, therefore, to increase the prevalence of these organisms in the soil and thus lead to increasingly severe losses from diseases. On the other hand, rotation of soybeans with other crops would tend to hold soil infestation to a minimum and thus lessen the losses caused by these diseases. For example, observations in Illinois show that when soybeans are grown on a field only once in 3 or 4 years, the severity of brown stem rot is much reduced (4).

FALL PLOWING

Soybean parasites that overwinter on or in diseased leaves and stems on the soil surface can be partially destroyed by thorough plowing under of this refuse after combining is completed in the fall. This practice should be particularly beneficial to growers who choose to grow soybeans continuously in the same fields rather than in a rotation.

DISEASE-FREE SEED

The parasites causing several of the diseases are known to infect the soybean seed and thus are spread into new fields and new localities. The bacterial diseases, pod and stem blight, frog-eye, downy mildew, and mosaic, are examples of diseases of this type. Therefore, no seed from fields in which any of these diseases were prevalent and destructive during the previous season should be planted.

SEED TREATMENT

Soybean seed treatment tests have been conducted at various locations in the United States since 1925 (42). More extensive tests have been made since 1940, and these have shown that the emergence of soybean seedlings in some districts can be increased 10 to 15 percent by treating the seed with a suitable chemical disinfectant before planting (74). However, there have been very few reports showing that an increased yield of soybeans resulted from the increased stand obtained by seed treatment. Apparently, the currently recommended seeding rate of 1 bushel or more per acre provides sufficient plants per acre for a maximum yield, even though 10 to 15 percent of the potential stand is lost as a result of seed decay and damping-off of seedlings. Consequently, soybean seed treatment has not been recommended as a general practice in the major producing States of the Corn Belt (5).

In the southern producing districts, on the other hand, the combination of poorer quality seed and cold, wet weather after planting sometimes results in the loss of 20 to

25 percent of the potential stand, if the soybean seed is not treated. Under such conditions, yield increases from seed treatment can be demonstrated, and the practice has been recommended in the South (41). A somewhat similar situation exists apparently in the northern limits of soybean cultivation (48).

Of the numerous seed-treating chemicals tested on soybeans, Arasan and Spergon dusts at the rate of 2 ounces per bushel have consistently given good results. The slurry formulations of these materials are apparently just as satisfactory as the dusts and are somewhat more convenient to use where slurry seed treaters are available. Tests conducted in Mississippi show that these treatments may be applied in the fall or early winter and, when so applied, will aid in maintaining the germinability of the planting seed during its storage period (41).

Observations made at numerous locations show that seed treatment with the above materials will have no serious effects on root nodulation in soils that have grown nodulated soybeans previously (5, 47, 72). In such fields, the plants will be inoculated with the soybean root nodule bacteria present in the soil from the previous crop.

When soybeans are being planted on land for the first time, the situation is somewhat different. Under these conditions, the seed should always be inoculated with a culture of the root-nodule-forming bacteria just before planting. The presence of a chemical disinfectant on the soybean seed at the time of inoculation impairs the efficiency of the inoculation process to a greater or lesser extent, depending upon the

disinfectant used. For example, mercurial compounds are extremely toxic to root nodule bacteria and should not be used on inoculated seeds. Some of the nonmercurial compounds show varying degrees of compatibility and may be used for seed treatment, especially if control of seedling diseases is more important than inoculation. Should a grower wish to practice both seed treatment and seed inoculation, the procedures should be in that order. Treating can be done at any time before planting, even in the preceding fall. Inoculating should be done just prior to planting, whether the seed is treated or not treated.

DUSTING

The great improvement in yield and quality resulting from timely applications of fungicidal dusts to the fruit and foliage of plants threatened with disease suggests the possibility of reducing losses from soybean diseases by similar methods. A project of this kind was initiated in North Carolina in 1944 and continued to 1950. Dust mixtures containing copper as the fungicide were used in all these years and in some years comparisons were made with other fungicides. Usually two or three varieties of soybeans differing in maturity dates and in susceptibility to diseases were included in the tests. The kind and severity of foliage diseases varied considerably from year to year, as influenced by prevailing weather conditions. Copper dust applications reduced bacterial and fungus foliar diseases in some years and thus apparently influenced the yield (56, 57, 60).

Yield increases ranging from 4 to 6 bushels per acre were obtained

from use of copper dusts in the years 1945, 1947, and 1949. No yield increases were obtained in 1946. Yields were depressed by dry weather in 1948 and only small increases were obtained. In 1950 relatively large increases were obtained on some varieties, but not on others. The mean yield increases obtained over the total period of the dusting tests have not been sufficient to pay for materials and labor of application when as many as five or six applications of 30 to 45 pounds of dust per acre were made. At present, dusting soybeans in the field for disease control does not appear to be an economical practice.

EXCLUSION OF FOREIGN DISEASES

While numerous soybean diseases are known to occur in the Oriental countries and in the United

States, there are others that are known to be prevalent only in the Orient. For example, a scab disease of soybean leaves, stems, and pods caused by a species of *Sphaceloma* was recognized in Japan for the first time in 1947 (40). So far this disease has not been reported in the United States. Soybean rust is another disease of this type. It is known to occur in Ceylon, China, India, and the Philippines, but it has not yet been found on soybeans in the United States (84). Constant vigilance on seed imports should be maintained, therefore, to prevent the introduction of diseases of this kind into the soybean-growing regions of the United States. Once introduced, these diseases will be here to stay, and in their new environment they may become major factors in soybean production.

LITERATURE CITED

- (1) ANONYMOUS.
1947. SOYBEAN. In *Canad. Plant Dis. Survey Ann. Rpt.* 26 (1946): 27-28.
- (2) ALLINGTON, W. B.
1945. WILDFIRE DISEASE OF SOYBEANS. *Phytopathology* 35: 857-869, illus.
- (3) ———
1946. BUD BLIGHT OF SOYBEAN CAUSED BY THE TOBACCO RING-SPOT VIRUS. *Phytopathology* 36: 319-322, illus.
- (4) ——— and CHAMBERLAIN, D. W.
1948. BROWN STEM ROT OF SOYBEAN. *Phytopathology* 38: 793-802, illus.
- (5) ——— KENT, G. C., TERVET, I. W., and KOEHLER, B.
1945. RESULTS OF THE UNIFORM SOYBEAN SEED TREATMENT TESTS IN 1944. U. S. Bur. Plant Indus., Soils, and Agr. Engin. Plant Dis. Rptr. Sup. 159: 220-224. [Processed.]
- (6) ARMSTRONG, G. M., and ARMSTRONG, J. K.
1950. BIOLOGICAL RACES OF THE FUSARIUM CAUSING WILT OF COWPEAS AND SOYBEANS. *Phytopathology* 40: 181-193, illus.
- (7) ARMSTRONG, J. K., and ARMSTRONG, G. M.
1951. PHYSIOLOGICAL RACES OF THE CROTALARIA WILT FUSARIUM. *Phytopathology* 41: 714-721, illus.
- (8) ATHOW, K., and PROBST, A. H.
1952. THE INHERITANCE OF RESISTANCE TO FROG-EYE LEAF SPOT OF SOYBEAN. *Phytopathology* 42: 660-662, illus.
- (9) ATKINS, J. G., Jr., and LEWIS, W. D.
1952. RHIZOCTONIA AERIAL BLIGHT OF SOYBEANS IN LOUISIANA. (Abstract) *Phytopathology* 42: 1.

- (10) BOENING, K.
1938. PHYLLOSTICTA - FLECKEN-KRANKHEIT DER SOJABOHNEN. Prakt. Bl. f. Pflanzenbau u. Pflanzenschutz. 16: 168-172, illus.
- (11) BOOSALIS, M. G.
1950. STUDIES ON THE PARASITISM OF RHIZOCTONIA SOLANI KUEHN ON SOYBEANS. Phytopathology 40: 820-831, illus.
- (12) CHAMBERLAIN, D. W.
1951. RESISTANCE TO BACTERIAL BLIGHT IN SOYBEANS. (Abstract) Phytopathology 41: 6.
- (13) ——— and KOEHLER, B.
1951. SOYBEAN DISEASES IN ILLINOIS. Ill. Agr. Expt. Sta. Cir. 676, 32 pp., illus.
- (14) CHITWOOD, B. G.
1949. ROOT-KNOT NEMATODES. PART I. A REVISION OF THE GENUS MELOIDOGYNE GOELDI, 1887. Helminthol. Soc. Wash. Proc. 16: 90-104, illus.
- (15) CLINTON, G. P.
1916. NOTES ON PLANT DISEASES OF CONNECTICUT. Conn. Agr. Expt. Sta. Ann. Rpt. (1915) 39: 421-451, illus.
- (16) COERPER, F. M.
1919. BACTERIAL BLIGHT OF SOYBEAN. Jour. Agr. Res. 18: 179-193, illus.
- (17) CONOVER, R. A.
1948. STUDIES OF TWO VIRUSES CAUSING MOSAIC DISEASES OF SOYBEANS. Phytopathology 38: 724-735, illus.
- (18) CRALL, J. M.
1948. DEFOLIATION OF SOYBEANS IN SOUTHEAST MISSOURI CAUSED BY PHYLLOSTICTA GLYCINUM. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 32: 184-186. [Processed.]
- (19) CRALLEY, E. M., and BEECHER, R. L.
1951. THE CONTROL OF SOYBEAN CHLOROSIS IN RICE-GROWING AREAS OF ARKANSAS BY POTASSIUM APPLICATIONS. (Abstract) Phytopathology 41: 7-8.
- (20) CROMWELL, R. O.
1917. FUSARIUM-BLIGHT, OR WILT DISEASE, OF THE SOYBEAN. Jour. Agr. Res. 8: 421-439, illus.
- (21) CROMWELL, R. O.
1919. FUSARIUM BLIGHT OF THE SOY BEAN AND THE RELATION OF VARIOUS FACTORS TO INFECTION. Nebr. Agr. Expt. Sta. Res. Bul. 14, 43 pp., illus.
- (22) DETURK, E. E.
1941. PLANT-NUTRIENT DEFICIENCY SYMPTOMS IN LEGUMES. In Hunger Signs in Crops. G. Hambidge, Ed. Pp. 241-266, illus. Washington, D. C.
- (23) EPPS, W. M., PATTERSON, J. C., and FREEMAN, I. E.
1951. PHYSIOLOGY AND PARASITISM OF SCLEROTIUM ROLFSSII. Phytopathology 41: 245-256, illus.
- (24) FEASTER, C. V.
1951. BACTERIAL PUSTULE DISEASE IN SOYBEANS: ARTIFICIAL INOCULATION, VARIETAL RESISTANCE, AND INHERITANCE OF RESISTANCE. Mo. Agr. Expt. Sta. Res. Bul. 487, 26 pp., illus.
- (25) GARDNER, M. W.
1927. INDIAN PLANT DISEASES. 1925. Ind. Acad. Sci. Proc. (1926) 36: 231-247, illus.
- (26) ——— and KENDRICK, J. B.
1921. SOYBEAN MOSAIC. Jour. Agr. Res. 22: 111-113, illus.
- (27) GEESEMAN, G. E.
1950. INHERITANCE OF RESISTANCE OF SOYBEANS TO PERONOSPORA MANSHURICA. Agron. Jour. 42: 608-613.
- (28) ———
1950. PHYSIOLOGIC RACES OF PERONOSPORA MANSHURICA ON SOYBEANS. Agron. Jour. 42: 257-258, illus.
- (29) GIBLER, J. W.
1951. PELLETING SOYBEAN SEED WITH FUNGICIDES FOR CONTROL OF DAMPING-OFF AND ROOT ROT. (Abstract) Phytopathology 41: 13-14.
- (30) GIBSON, F.
1922. SUNBURN AND APHID INJURY OF SOYBEANS AND COWPEAS. Ariz. Agr. Expt. Sta. Tech. Bul. 2, 41-46, illus.
- (31) GRAHAM, J. H.
1951. OVERWINTERING AND ASSOCIATION OF THREE BACTERIAL PATHOGENS OF SOYBEAN. (Abstract) Phytopathology 41: 14.

- (32) HARTWIG, E. E., and JOHNSON, H. W.
1953. EFFECT OF THE BACTERIAL PUSTULE DISEASE ON YIELD AND CHEMICAL COMPOSITION OF SOYBEANS. *AGRON. JOUR.* 45: 22-23.
- (33) ——— and LEHMAN, S. G.
1951. INHERITANCE OF RESISTANCE TO THE BACTERIAL PUSTULE DISEASE IN SOYBEANS. *AGRON. JOUR.* 43: 226-229.
- (34) HEDGES, F.
1924. A STUDY OF BACTERIAL PUSTULE OF SOYBEAN, AND A COMPARISON OF BACT. PHASEOLI SOJENSE HEDGES WITH BACT. PHASEOLI EFS. *JOUR. AGR. RES.* 29: 229-251, illus.
- (35) HEINZE, K., and KOEHLER, E.
1941. DIE MOSAIKKRANKHEIT DER SOJABOHNE UND IHRE UEBERTRAGUNG DURCH INSEKTEN. *PHYTOPATH. ZTSCHR.* 13: [207]-242, illus.
- (36) HIGGINS, B. B.
1927. PHYSIOLOGY AND PARASITISM OF SCLEROTIUM ROLFII SACC. *PHYTOPATHOLOGY* 17: 417-448, illus.
- (37) HILDEBRAND, A. A., and KOCH, L. W.
1951. A STUDY OF SYSTEMIC INFECTION BY DOWNY MILDEW OF SOYBEAN WITH SPECIAL REFERENCE TO SYMPTOMATOLOGY, ECONOMIC SIGNIFICANCE AND CONTROL. *SCI. AGR.* 31: 505-518, illus.
- (38) ——— and KOCH, L. W.
1952. OBSERVATIONS ON A ROOT AND STEM ROT OF SOYBEANS NEW TO ONTARIO, CAUSED BY PYTHIUM ULTIMUM TROW. *SCI. AGR.* 32: 574-580, illus.
- (39) HOLDEMAN, Q. L.
1950. SOME FALCATE-SPORED COLLETOTRICHUMS ON LEGUMES. (Abstract) *PHYTOPATHOLOGY* 40: 12-13.
- (40) JENKINS, A. E.
1951. SPHACELOMA SCAB, A NEW DISEASE OF SOYBEANS DISCOVERED BY PLANT PATHOLOGISTS IN JAPAN. *U. S. BUR. PLANT INDUS., SOILS, and AGR. ENGIN., PLANT DIS. RPTR.* 35: 110-111, illus. [Processed.]
- (41) JOHNSON, H. W.
1951. SOYBEAN SEED TREATMENT. *Soybean Digest* 11 (7): 17-19, illus.
- (42) ——— and KOEHLER, B.
1943. SOYBEAN DISEASES AND THEIR CONTROL. *U. S. DEPT. AGR. FARMERS' BUL.* 1937, 24 pp., illus.
- (43) ——— and LEFEBVRE, C. L.
1942. DOWNY MILDEW ON SOYBEAN SEEDS. *U. S. BUR. PLANT INDUS., PLANT DIS. RPTR.* 26: 49-50. [Processed.]
- (44) JONES, F. R., and TORRIE, J. H.
1946. SYSTEMIC INFECTION OF DOWNY MILDEW IN SOYBEAN AND ALFALFA. *PHYTOPATHOLOGY* 36: 1057-1059, illus.
- (45) KENDRICK, J. B., and GARDNER, M. W.
1921. SEED TRANSMISSION OF SOYBEAN BACTERIAL BLIGHT. *PHYTOPATHOLOGY* 11: [340]-342, illus.
- (46) ——— and GARDNER, M. W.
1924. SOYBEAN MOSAIC: SEED TRANSMISSION AND EFFECT ON YIELD. *JOUR. AGR. RES.* 27: 91-98.
- (47) KERNEKAMP, M. F.
1948. CHEMICAL TREATMENT OF SOYBEAN SEED IN RELATION TO NODULATION BY NODULE BACTERIA. *PHYTOPATHOLOGY* 38: 955-959.
- (48) ———
1948. SHOULD SOYBEAN SEED BE TREATED? *MINN. FARM and HOME SCI.* 5 (3): 5.
- (49) ——— and GIBLER, J. W.
1951. RESISTANCE IN SOYBEANS TO ROOT ROT CAUSED BY RHIZOCTONIA SOLANI. (Abstract) *PHYTOPATHOLOGY* 41: 21.
- (50) LARSH, H. W.
1944. SUMMARY REPORT OF PLANT DISEASES IN OKLAHOMA, 1943. *U. S. BUR. PLANT INDUS., SOILS, and AGR. ENGIN., PLANT DIS. RPTR. SUPP.* 149: 317-326. [Processed.]
- (51) LEHMAN, S. G.
1923. POD AND STEM BLIGHT OF SOYBEAN. *MO. BOT. GARD. ANN.* 10: 111-178, illus.

- (52) LEHMAN, S. G.
1928. FROG-EYE LEAF SPOT OF SOYBEAN CAUSED BY *CERCOSPORA DIAZU* MIURA. Jour. Agr. Res. 36: 811-833, illus.
- (53) ———
1931. POWDERY MILDEW OF SOYBEAN. Elisha Mitchell Sci. Soc. Jour. 46: 190-195, illus.
- (54) ———
1934. FROG-EYE (*CERCOSPORA DIAZU* MIURA) ON STEMS, PODS, AND SEEDS OF SOYBEAN, AND THE RELATION OF THESE INFECTIONS TO RECURRENCE OF THE DISEASE. Jour. Agr. Res. 48: 131-147, illus.
- (55) ———
1943. OCCURRENCE OF YEAST SPOT ON SOYBEAN IN NORTH CAROLINA. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 27: 602. [Processed.]
- (56) ———
1944. DUSTING SOYBEANS FOR CONTROL OF BACTERIAL PUSTULE. (Abstract) Phytopathology 34: 1007-1008.
- (57) ———
1946. CONTROL OF BACTERIAL PUSTULE OF SOYBEAN BY DUSTING. (Abstract) Phytopathology 36: 405.
- (58) ———
1947. POWDERY MILDEW OF SOYBEAN. (Abstract) Phytopathology 37: 434.
- (59) ———
1950. PURPLE STAIN OF SOYBEAN SEEDS. N. C. Agr. Expt. Sta. Bul. 369, 11 pp., illus. and GRAHAM, J. H.
- (60) ———
1948. RESULTS FROM DUSTING SOYBEANS WITH COPPER IN 1947. (Abstract) Phytopathology 38: 570.
- (61) ———
MURAKISHI, H., and GRAHAM, J. H.
1951. A LEAF SPOT OF SOYBEAN CAUSED BY *SCLEROTIUM ROLFII*. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 35: 167-168, illus. [Processed.]
- (62) LEHMAN, S. G., and WOLF, F. A.
1926. *PYTHIUM* ROOT ROT OF SOYBEAN. Jour. Agr. Res. 33: 375-380, illus.
- (63) ——— and WOLF, F. A.
1926. SOYBEAN ANTHRACNOSE. Jour. Agr. Res. 33: 381-390, illus.
- (64) LING, L.
1951. BIBLIOGRAPHY OF SOYBEAN DISEASES. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. Sup. 204: 110-173. [Processed.]
- (65) LUTTRELL, E. S.
1947. *DIAPORTE PHASEOLORUM* VAR. *SOJAE* ON CROP PLANTS. Phytopathology 37: 445-465, illus.
- (66) MURAKISHI, H. H.
1951. PURPLE SEED STAIN OF SOYBEAN. Phytopathology 41: 305-318, illus.
- (67) OLIVE, L. S.
1949. TARGET SPOT OF COWPEA AND SOYBEAN. Mycologia 41: 355.
- (68) ——— BAIN, D. C., and LEFEBVRE, C. L.
1945. A LEAFSPOT OF COWPEA AND SOYBEAN CAUSED BY AN UNDESCRIBED SPECIES OF *HELMINTHOSPORIUM*. Phytopathology 35: 822-831, illus.
- (69) ——— and WALKER, E. A.
1944. A SEVERE LEAFSPOT OF SOYBEAN CAUSED BY *PHYLLISTICTA SOJAEICOLA*. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 28: 1122-1123. [Processed.]
- (70) SAMSON, R. W.
1942. TOBACCO RING-SPOT ON EDIBLE SOYBEANS IN INDIANA IN 1941. U. S. Bur. Plant Indus., Plant Dis. Rptr. 26: 382. [Processed.]
- (71) SASSER, J. N.
1952. IDENTIFICATION OF ROOT-KNOT NEMATODES (*MELOIDOGYNE* SPP.) BY HOST REACTIONS. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 36: 84-86, illus. [Processed.]

- (72) SHERF, A. F., and REDDY, C. S.
1952. SEED TREATMENT AND INOCULATION STUDIES ON SOYBEANS IN IOWA FIELDS, 1950-1951. (Abstract) *Phytopathology* 42: 19.
- (73) SHERWIN, H. S., and KREITLOW, K. W.
1952. DISCOLORATION OF SOYBEAN SEEDS BY THE FROGEYE FUNGUS, *CERCOSPORA SOJINA*. *Phytopathology* 42: 568-572, illus.
- (74) ——— LEBEVRE, C. L., and LEUKEL, R. W.
1948. EFFECT OF SEED TREATMENT ON THE GERMINATION OF SOYBEANS. *Phytopathology* 38: 197-204, illus.
- (75) TEHON, L. R., and DANIELS, E. Y.
1927. NOTES ON THE PARASITIC FUNGI OF ILLINOIS III. *Mycologia* 19: 110-129, illus.
- (76) TIFFANY, L. H.
1951. DELAYED SPORULATION OF *COLLETOTRICHUM* ON SOYBEAN. *Phytopathology* 41: 975-985, illus.
- (77) TIMNICK, M. B., LILLY, V. G., and BARNETT, H. L.
1951. FACTORS AFFECTING SPORULATION OF *DIAPORTHE PHASEOLORUM* VAR. *BATATATIS* FROM SOYBEAN. *Phytopathology* 41: 327-336, illus.
- (78) WALTERS, H. J.
1952. SOME RELATIONSHIPS OF THREE PLANT VIRUSES TO THE DIFFERENTIAL GRASSHOPPER, *MELANOPLUS DIFFERENTIALIS* (THOS.). *Phytopathology* 42: 355-362, illus.
- (79) WEI, C. T.
1950. NOTES ON *CORYNESPORA*. *Commonw. Mycol. Inst. Mycol. Papers* 34, 10 pp., illus.
- (80) WEIMER, J. L.
1950. BLACKPATCH OF SOYBEAN AND OTHER FORAGE LEGUMES. *Phytopathology* 40: 782-784, illus.
- (81) WEISS, F.
1946. CHECK LIST REVISION. U. S. Bur. Plant Indus., Soils, and Agr. Engin., Plant Dis. Rptr. 30: 130-137. [Processed.]
- (82) WELCH, A. W., and GILMAN, J. C.
1948. HETERO- AND HOMO-THALIC TYPES OF *DIAPORTHE* ON SOYBEANS. *Phytopathology* 38: 628-637, illus.
- (83) WOLF, F. A., and LEHMAN, S. G.
1926. BROWN-SPOT DISEASE OF SOY BEAN. *Jour. Agr. Res.* 33: 365-374, illus.
- (84) ——— and LEHMAN, S. G.
1926. DISEASES OF SOY BEANS WHICH OCCUR BOTH IN NORTH CAROLINA AND THE ORIENT. *Jour. Agr. Res.* 33: 391-396.

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